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Editorial Comment

Coronary Plaque Morphology in Postinfarction Patients: Implications for Early Versus Deferred Coronary Angioplasty*

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The present study. The study of Davies et al. (1) in this issue of the Journal is a detailed angiographic analysis that again confirms the relation of acute myocardial infarction to unstable plaque and to certain morphologic features. The importance of this communication is the demonstration that the angiographic correlates of the unstable plaque and associated thrombus improve or disappear with time and anticoagulant therapy. The authors (1) extend previous reports (2–4) by showing changes in lesion morphology that occur within 2 weeks after thrombolytic therapy. Nakagawa et al. (3) earlier reported lesion morphologic features in 43 consecutive patients immediately after thrombolytic therapy and after 1 month of anticoagulant therapy. A change in lesion classification occurred in 19 (44%) of 43 patients. Similarly, Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) investigators (4) noted that 14% of 98 patients, observed to have critical stenosis at 90 min after thrombolytic therapy, had hemodynamically insignificant stenosis after 1 week. The demonstration of these morphologic changes after thrombolytic therapy has obvious implications for applying coronary angioplasty and other revascularization strategies in post–myocardial infarction patients.

Immediate coronary angioplasty after thrombolytic therapy not only exposes some patients to the risk of an unnecessary procedure, but the results of the TAMI, Thrombolysis in Myocardial Infarction Phase IIA (TIMI-IIA) and European Cooperative Study Group trials all show that very early angioplasty was associated with increased mortality, abrupt closure and emergency bypass surgery rates compared with deferred angioplasty. The ideal time to electively intervene with angioplasty after acute myocardial infarction is not known. This report by Davies et al. (1) indicates that the culprit lesion at a mean interval of 3.3 days after infarction frequently has unstable characteristics and that angiographic indexes of instability begin to regress 2 to 8 days later. Exactly how this translates in terms of angioplasty success and complication rates must be determined. Angioplasty in the presence of thrombus has a higher complication rate in our hands in the patient with acute infarction or unstable angina. In a series of patients with a large intracoronary thrombus we noted resolution or significant regression of thrombus in most patients after 1 week of heparin and aspirin therapy (5). Coronary angioplasty was subsequently performed in those patients with persisting stenosis without increased complications. Thus, we believe prolonged heparinization was beneficial. Mooney et al. (6), in a large series of patients who underwent angioplasty despite intracoronary thrombus, reported more frequent in-hospital chest pain and a 7% incidence rate of in-hospital coronary bypass surgery for unsuccessful coronary angioplasty but no deaths and only one Q wave infarction.

Implications for timing of angioplasty after infarction. An important practical consideration is whether the improved result obtained with deferred angioplasty in the setting of myocardial infarction or unstable angina is due to resolution of thrombus alone or whether some further stabilization in the activity of the underlying plaque is also helpful. If the former is true, then a more rapid resolution of the thrombus might be achieved with additional thrombolytic therapy probably followed by angioplasty. On the other hand, if inactivation of the plaque to include alterations in its morphology, its cellular composition including inflammatory cells and its biochemistry are important, then a period of stabilization with time and anticoagulant therapy may be essential. Definitive answers to these questions could come only from prospective randomized trials that compare patients with unstable morphology treated immediately after thrombolytic therapy and after anticoagulant therapy with a time delay. Davies et al. (1) are obviously convinced, as are we, that the strategy of performing coronary angioplasty after the resolution of thrombus or stabilization of the plaque, or both, is superior to very early intervention that seems to lead to more complications.

However, it should not be assumed that this strategy will remain the preferred one. Advances in stabilizing lesions and in preventing thrombosis are undergoing active investigation. Synthetic antithrombins can inactivate thrombotic surfaces in experimental animals, powerful antiplatelet agents are under investigation and heparin fragments have been shown to significantly blunt the phenotypic transformation of the smooth muscle cells. Other methods for controlling cellular responses within unstable lesions may prove suc-

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cessful. If these strategies prove effective and practical in humans, then acute stabilization of the plaque may provide an optimal substrate for coronary angioplasty. Until such strategies are proved, watchful waiting with anticoagulation provides a reasonable strategy for patients who can be stabilized and followed. For patients whose lesions become unstable, coronary angioplasty still remains a viable alternative.

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